

# William R Sellers

## List of Publications by Year in descending order

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Version: 2024-02-01

45  
papers

20,630  
citations

117619

34  
h-index

233409

45  
g-index

50  
all docs

50  
docs citations

50  
times ranked

34627  
citing authors

#	ARTICLE	IF	CITATIONS
1	David Livingston (1941–2021). <i>Molecular Cell</i> , 2022, 82, 4-7.	9.7	2
2	Comparative optimization of combinatorial CRISPR screens. <i>Nature Communications</i> , 2022, 13, 2469.	12.8	13
3	Distinct CDK6 complexes determine tumor cell response to CDK4/6 inhibitors and degraders. <i>Nature Cancer</i> , 2021, 2, 429-443.	13.2	29
4	Targeting pan-essential genes in cancer: Challenges and opportunities. <i>Cancer Cell</i> , 2021, 39, 466-479.	16.8	88
5	Molecular basis for substrate recruitment to the PRMT5 methylosome. <i>Molecular Cell</i> , 2021, 81, 3481-3495.e7.	9.7	41
6	Paralog knockout profiling identifies DUSP4 and DUSP6 as a digenic dependence in MAPK pathway-driven cancers. <i>Nature Genetics</i> , 2021, 53, 1664-1672.	21.4	61
7	Are CRISPR Screens Providing the Next Generation of Therapeutic Targets?. <i>Cancer Research</i> , 2021, 81, 5806-5809.	0.9	7
8	A Proof of Concept for Biomarker-Guided Targeted Therapy against Ovarian Cancer Based on Patient-Derived Tumor Xenografts. <i>Cancer Research</i> , 2020, 80, 4278-4287.	0.9	12
9	Quantitative Proteomics of the Cancer Cell Line Encyclopedia. <i>Cell</i> , 2020, 180, 387-402.e16.	28.9	596
10	FGF401, A First-In-Class Highly Selective and Potent FGFR4 Inhibitor for the Treatment of FGF19-Driven Hepatocellular Cancer. <i>Molecular Cancer Therapeutics</i> , 2019, 18, 2194-2206.	4.1	65
11	GEMINI: a variational Bayesian approach to identify genetic interactions from combinatorial CRISPR screens. <i>Genome Biology</i> , 2019, 20, 137.	8.8	30
12	Metabolomic adaptations and correlates of survival to immune checkpoint blockade. <i>Nature Communications</i> , 2019, 10, 4346.	12.8	139
13	Capmatinib (INC280) Is Active Against Models of Non-Small Cell Lung Cancer and Other Cancer Types with Defined Mechanisms of MET Activation. <i>Clinical Cancer Research</i> , 2019, 25, 3164-3175.	7.0	104
14	Next-generation characterization of the Cancer Cell Line Encyclopedia. <i>Nature</i> , 2019, 569, 503-508.	27.8	2,149
15	The landscape of cancer cell line metabolism. <i>Nature Medicine</i> , 2019, 25, 850-860.	30.7	350
16	The potent and selective cyclin-dependent kinases 4 and 6 inhibitor ribociclib (LEE011) is a versatile combination partner in preclinical cancer models. <i>Oncotarget</i> , 2018, 9, 35226-35240.	1.8	59
17	Dose and Schedule Determine Distinct Molecular Mechanisms Underlying the Efficacy of the p53-MDM2 Inhibitor HDM201. <i>Cancer Research</i> , 2018, 78, 6257-6267.	0.9	60
18	Resistance mechanisms to TP53-MDM2 inhibition identified by in vivo piggyBac transposon mutagenesis screen in an Arf <sup>fl/fl</sup> mouse model. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, 3151-3156.	7.1	48

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19	Discovery and Optimization of HKT288, a Cadherin-6 Targeting ADC for the Treatment of Ovarian and Renal Cancers. <i>Cancer Discovery</i> , 2017, 7, 1030-1045.	9.4	40
20	Project DRIVE: A Compendium of Cancer Dependencies and Synthetic Lethal Relationships Uncovered by Large-Scale, Deep RNAi Screening. <i>Cell</i> , 2017, 170, 577-592.e10.	28.9	506
21	Combined ALK and MDM2 inhibition increases antitumor activity and overcomes resistance in human ALK mutant neuroblastoma cell lines and xenograft models. <i>ELife</i> , 2017, 6, .	6.0	35
22	ERG signaling in prostate cancer is driven through PRMT5-dependent methylation of the Androgen Receptor. <i>ELife</i> , 2016, 5, .	6.0	64
23	Allosteric Inhibition of SHP2: Identification of a Potent, Selective, and Orally Efficacious Phosphatase Inhibitor. <i>Journal of Medicinal Chemistry</i> , 2016, 59, 7773-7782.	6.4	229
24	High-Order Drug Combinations Are Required to Effectively Kill Colorectal Cancer Cells. <i>Cancer Research</i> , 2016, 76, 6950-6963.	0.9	30
25	CRISPR Screens Provide a Comprehensive Assessment of Cancer Vulnerabilities but Generate False-Positive Hits for Highly Amplified Genomic Regions. <i>Cancer Discovery</i> , 2016, 6, 900-913.	9.4	320
26	Allosteric inhibition of SHP2 phosphatase inhibits cancers driven by receptor tyrosine kinases. <i>Nature</i> , 2016, 535, 148-152.	27.8	674
27	Disordered methionine metabolism in MTAP/CDKN2A-deleted cancers leads to dependence on PRMT5. <i>Science</i> , 2016, 351, 1208-1213.	12.6	374
28	Oncogene addiction: pathways of therapeutic response, resistance, and road maps toward a cure. <i>EMBO Reports</i> , 2015, 16, 280-296.	4.5	200
29	Studying clonal dynamics in response to cancer therapy using high-complexity barcoding. <i>Nature Medicine</i> , 2015, 21, 440-448.	30.7	408
30	High-throughput screening using patient-derived tumor xenografts to predict clinical trial drug response. <i>Nature Medicine</i> , 2015, 21, 1318-1325.	30.7	1,065
31	A distinct p53 target gene set predicts for response to the selective p53 HDM2 inhibitor NVP-CGM097. <i>ELife</i> , 2015, 4, .	6.0	65
32	Inhibiting Tankyrases Sensitizes KRAS-Mutant Cancer Cells to MEK Inhibitors via FGFR2 Feedback Signaling. <i>Cancer Research</i> , 2014, 74, 3294-3305.	0.9	34
33	Modelling vemurafenib resistance in melanoma reveals a strategy to forestall drug resistance. <i>Nature</i> , 2013, 494, 251-255.	27.8	665
34	Rescue Screens with Secreted Proteins Reveal Compensatory Potential of Receptor Tyrosine Kinases in Driving Cancer Growth. <i>Cancer Discovery</i> , 2012, 2, 948-959.	9.4	94
35	FGFR Genetic Alterations Predict for Sensitivity to NVP-BCJ398, a Selective Pan-FGFR Inhibitor. <i>Cancer Discovery</i> , 2012, 2, 1118-1133.	9.4	297
36	Modulation of Activation-Loop Phosphorylation by JAK Inhibitors Is Binding Mode Dependent. <i>Cancer Discovery</i> , 2012, 2, 512-523.	9.4	106

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37	The Cancer Cell Line Encyclopedia enables predictive modelling of anticancer drug sensitivity. <i>Nature</i> , 2012, 483, 603-607.	27.8	6,473
38	A Drug Resistance Screen Using a Selective MET Inhibitor Reveals a Spectrum of Mutations That Partially Overlap with Activating Mutations Found in Cancer Patients. <i>Cancer Research</i> , 2011, 71, 5255-5264.	0.9	109
39	The landscape of somatic copy-number alteration across human cancers. <i>Nature</i> , 2010, 463, 899-905.	27.8	3,331
40	COT drives resistance to RAF inhibition through MAP kinase pathway reactivation. <i>Nature</i> , 2010, 468, 968-972.	27.8	1,325
41	Inhibition of Hsp90 Down-regulates Mutant Epidermal Growth Factor Receptor (EGFR) Expression and Sensitizes EGFR Mutant Tumors to Paclitaxel. <i>Cancer Research</i> , 2008, 68, 589-596.	0.9	172
42	Inclusion of the <i>ASH1</i> gene that governs the neuroendocrine differentiation of lung epithelium as an additional prototypic 'lineage-survival oncogene'. <i>Nature Reviews Cancer</i> , 2007, 7, 68-68.	28.4	0
43	Frequent HIN-1 Promoter Methylation and Lack of Expression in Multiple Human Tumor Types. <i>Molecular Cancer Research</i> , 2004, 2, 489-494.	3.4	46
44	The EZH2 polycomb transcriptional repressorâ€”a marker or mover of metastatic prostate cancer?. <i>Cancer Cell</i> , 2002, 2, 349-350.	16.8	86
45	Cyclin D1 suppresses retinoblastoma protein-mediated inhibition of TAFII250 kinase activity. <i>Oncogene</i> , 2000, 19, 5703-5711.	5.9	21